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The Stretch Reflex and H-Reflex of the Human Soleus Muscle During Walking

Jacob Buus Andersen and Thomas Sinkjær

Due to the complexity of applying a well-defined stretch during human walking, most of our knowledge about the short latency stretch reflex modulation in humans is based on H-reflex studies. To illuminate the difference between the two methodologies, both types of reflexes were evoked in the same subjects, same experiment. Stretch reflexes were evoked via a stretch device capable of evoking stretch reflexes of the human soleus muscle during walking. H-reflexes were elicited by an electrical stimulation of the tibial nerve at the popliteal fossa at the knee. A significantly different modulation of the two reflexes was found in the late stance where the stretch reflex decreased in relation to the H-reflex. This was consistent with an unloading of the muscle spindles during the push-off in late stance, suggesting a complex alpha-gamma coactivation, if any, at this time of the step. The soleus stretch reflex and H-reflex were compared during the stance phase of walking and sitting at matched soleus EMG activity. No difference was found in the amplitude of the stretch reflex. However, there was a significant decrease of the H-reflex during the stance phase of walking, consistent with a task-specific presynaptic mediated reflex control. It is proposed that the short latency stretch reflex during walking is not sensitive to such a presynaptic inhibition.

Studies in humans have demonstrated marked differences in the size of the soleus H-reflex during standing and walking at matched levels of soleus EMG activity, likely explained by a task-dependent presynaptic inhibition of the Group Ia afferents (Capaday & Stein, 1987; Edamura et al., 1991; Faist et al., 1996). Most of our knowledge about the short latency stretch reflex during walking is based on H-reflex studies, which has led to the belief that the short latency stretch reflex is regulated in much the same way as the H-reflex (e.g., Capaday & Stein, 1986; Stein et al., 1993). However, an increasing number of H- and stretch reflex studies have shown that this might not be correct (Morita et al., 1998; Nielsen & Petersen, 1994; Sinkjær et al., 1996). Sinkjær et al. (1996) showed that the size of the short latency soleus stretch reflex was of equal strength during standing and walking at matched levels of soleus EMG activity, suggesting that the short latency stretch reflex during walking is not sensitive to presynaptic inhibition. However, the H- and stretch reflex have not been investigated during walking within

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the same subjects, and thus no conclusive studies are available on the differences, if any, in the stretch and H-reflex during walking. The present study investigated the two reflexes in the soleus muscle in the same subjects within the same experiment, and reflex amplitudes were compared at carefully matched levels of soleus EMG activity and walking speed.

Method

Protocol

The experiments were performed with 8 healthy subject (6 M, 2 F; ages 22–32 years). Bipolar EMG electrodes were placed 2 cm apart on the left leg—on the soleus muscle and on the anterior tibialis. A ground electrode was placed under the knee. The EMGs were amplified and filtered from 20 to 1000 Hz (DISA, Model 15C01).

Stretch reflexes were elicited by a portable stretch device capable of rotating the human ankle joint during walking on a treadmill (Andersen & Sinkjær, 1995). The system consisted of a mechanical joint, which was mounted level with the ankle joint. The mechanical joint was connected to a powerful actuator system by means of two flexible bowden wires. Plaster casts were made in polypropylene to give a unique interface from the mechanical joint to the subject's ankle.

A heel contact was placed in the subject's left shoe, and an insole was put in the right shoe to match the change in height caused by the casting in the left shoe. The subject walked with a natural cadence at 3.5–4 km/hr. After an adaptation period of 5 min, an EMG profile, triggered from heel contact to heel contact, was performed on the soleus and tibialis anterior based on 20 steps. The step cycle was divided into 10 segments and an 8° stretch was imposed. The stretches were randomly imposed between the segments with an interval of 4–6 steps apart until 8–10 stretches were accomplished in each time segment. The procedure was repeated with the electrically evoked H-reflex. To evoke H-reflexes, a spring-loaded ball electrode was placed in the popliteal fossa with a reference electrode located above the patella. The M-wave was held at a constant level during walking. The H-reflex experiment was performed without removing the portable stretch apparatus.

After walking, the subjects were seated on a chair with the left shoe fastened to the floor. They were then told to do isometric contractions of the soleus EMG. This corresponded to the level of walking in the midstance phase. The subjects were given visual feedback of a rectified, low-pass filtered (1st order 2 Hz) soleus EMG at a monitor. They were told to perform the contraction until 10 trials of stretch reflexes and 10 trials of H-reflexes were averaged with an interval of about 4 sec between each stimulus.

Data Analysis

The data were analyzed on a computer as follows. The EMG activity of the soleus muscle was averaged, rectified, and low-pass filtered at 20 Hz (1st order). The reflex amplitude was measured as the peak amplitude minus an average background activity of 30 ms before the onset of the reflex response.

The data were tested by use of the Wilcoxon matched-pair test with a level of significance of $p < 0.05$.

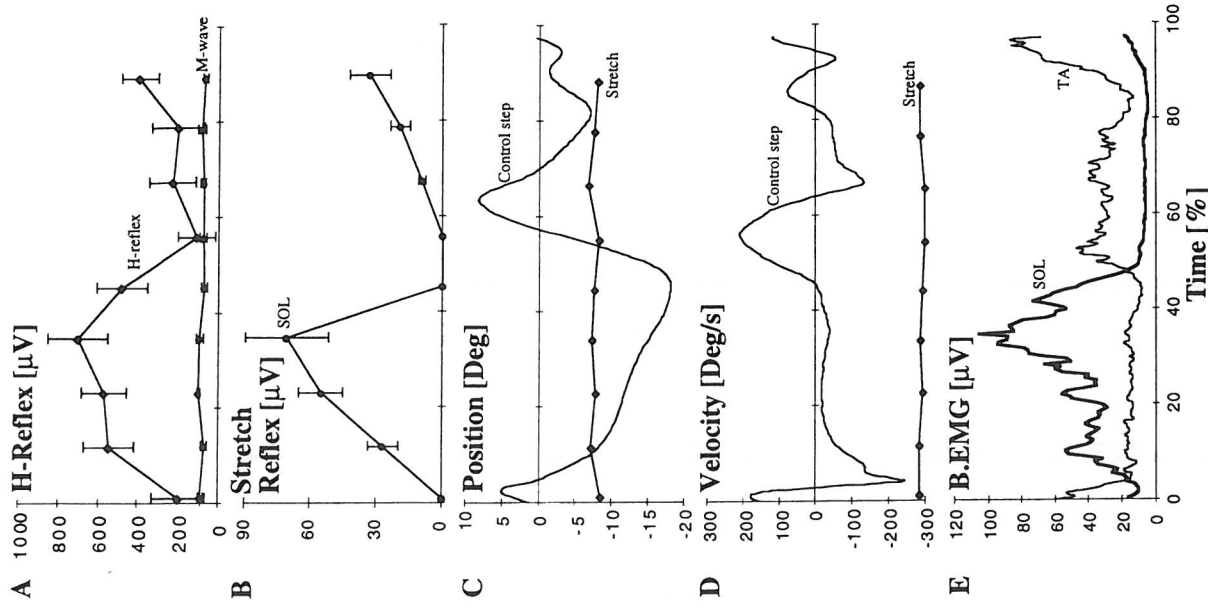


Figure 1 — Example of stretch and H-reflex modulation during gait cycle in one subject (DM) walking at 3.8 km/h. Gait cycle divided into 10 segments: (A) H-reflex modulation in the soleus; M-wave held at a constant level of 8–12% of M-max. (B) Soleus stretch reflex. (C) Position of ankle joint in control steps; stretch perturbation was kept at a constant level with displacement of 8°. (D) Velocity of ankle joint during control steps; stretch perturbation was kept at a constant level with displacement velocity of 280°/sec. (E) Background EMG of soleus (thick line) and tibialis anterior (thin line).

Results

The soleus stretch reflex was evoked by a stretch of $8.06 \pm 0.05^\circ$ and with a velocity of $298 \pm 2.46^\circ/\text{s}$ ($N = 8$). The stretch resulted in a stretch reflex response with a peak latency of 56.0 ± 0.7 ms measured from stretch onset. The soleus H-reflex was elicited at a fixed percent of M-max. The peak latency was 39.5 ± 0.7 ms measured from stimulation onset.

Figure 1 shows an example (Subject DM) of the modulation of the stretch reflex in the soleus compared to the soleus H-reflex. The soleus H-reflex increased until midstance, decreased to a level of the M-wave at the transition from stance to swing, and again slowly increased toward the end of the swing phase. The M-wave was held at a constant level of 8–12% of M-max (Figure 1A). The soleus stretch reflex built up in the stance phase and was completely inhibited in the transition from stance to swing. Again it slowly increased during the swing phase (Figure 1B). Figures 1C and 1D illustrate the position and the derived velocity. At the initial part of the stance phase just after foot-flat and toe-off in the swing phase, a fast dorsiflexion was seen at the minimum points of the stretch reflex. Throughout the gait cycle, the stretch amplitude and velocity were kept at a constant level. The background soleus and tibialis anterior EMG are depicted during a control step in Figure 1E.

Figure 2 shows the stretch reflex and H-reflex for all subjects. The data were normalized to the maximal value for each subject and then averaged. A significant difference of the two reflexes was not found, except for two points at the end of stance phase. The normalized averaged soleus EMG in the control step is shown in Figure 2B.

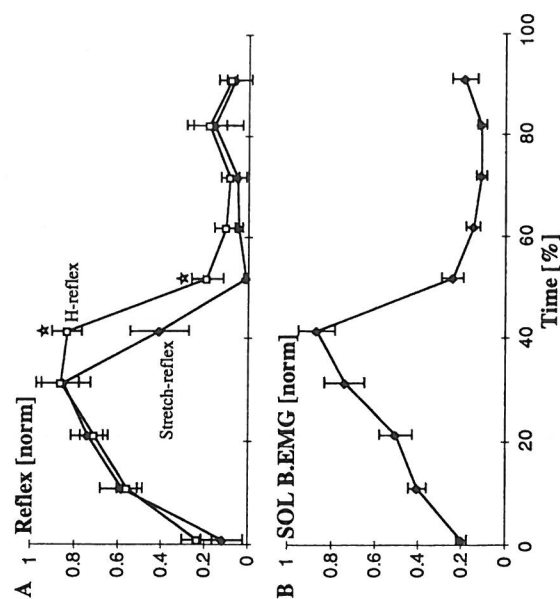


Figure 2 — H-reflex and stretch reflex modulation in all 8 subjects. (A) Averaged stretch reflex modulation and H-reflex modulation, each normalized to maximal value of individual subjects. Star indicates significant difference between both reflexes. (B) Averaged soleus background EMG normalized to maximal value of each subject.

Seven sitting subjects were asked to perform an isometric contraction which matched the soleus EMG level during the midstance phase of walking. The amplitude of the H-reflex was significantly decreased ($p < 0.01$) from sitting ($642.9 \pm 309.2 \mu\text{V}$) to walking condition ($223.0 \pm 64.7 \mu\text{V}$) whereas the stretch reflex was unchanged ($p > 0.05$) from sitting ($63.7 \pm 15.7 \mu\text{V}$) to walking ($66.2 \pm 3.6 \mu\text{V}$).

Discussion

Comparing the H- and stretch reflex at matched conditions within the same subjects, the present study showed that (a) the two reflexes were modulated alike during the step, except in the late stance phase where the stretch reflex was proportionately lower than the H-reflex; and (b) in midstance the amplitude of the stretch reflex was the same during walking and sitting at matched levels of EMG activity. In contrast, the H-reflex amplitude decreased in strength during walking as compared to sitting.

In late stance, the stretch reflex decreased as the soleus EMG activity increased (Figure 2). At this time of the step the muscle fibers have started to shorten due to the marked increase in the soleus alpha-motoneuron activity (Hoffer et al., 1989; Voigt et al., 1996). If the gamma-motoneurons do not compensate for this unloading of intrafusal muscle fibers, the afferent response from the muscle spindles to a stretch will decrease, further decreasing the stretch reflex. This suggests either a complex alpha-gamma coactivation during human walking as previously described in cats (Bessou et al., 1990), or no alpha-gamma coactivation at this time of the step. In contrast to the stretch reflex, the H-reflex remains at a high level as the alpha-motoneuron excitability increases at this time in the stance phase.

Another explanation of the decreased stretch reflex in the late stance could be an absorption of the imposed stretch into the passive tissue due to the high stiffness of the muscle fibers at this point. This will lead to a smaller stretch of the intrafusal fibers, which again decreases the stretch reflex. However, stretches imposed during tonic contractions at comparable EMG levels in sitting human subjects do not show a decrease in the soleus stretch reflex at increasing contraction levels (e.g., Toft et al., 1991), suggesting that the imposed stretch is transmitted to the muscle also at high contraction levels.

We found a marked decrease in the size of the soleus H-reflex during walking as compared to sitting at matched levels of soleus EMG activity. This is consistent with the task-specific presynaptic mediated reflex control as proposed by Capaday and Stein (1986). In contrast to this, no difference was found between the soleus stretch reflex during the stance phase of walking and sitting at the exact same conditions as investigated for the H-reflex. Several interpretations might explain these differences between the stretch and H-reflex:

1. The stimuli evoking the two reflexes (electrical stimulus and stretch) are likely to activate a different number of Ia afferents, Group II afferents, and cutaneous afferents. Also, the H-reflex method causes a very narrow and synchronized Ia afferent inflow to the homonymous motoneuron pool whereas the mechanical stretch causes a less synchronized afferent input to the homonymous motoneuron pool due to its duration and shape.
2. The H-reflex reflects the central gain of the reflex arc, whereas the stretch reflex is also determined by the sensitivity of the muscle spindles. When a

voluntary activation of the muscles is made, clear signs of accompanying fusimotor activation are found: increased spindle afferent firing in isometric contractions, maintained firing during muscle shortening, and increased firing during muscle lengthening (Prochazka 1995). These changes are not reflected in the H-reflex.

3. When the ankle extensors are stretched, there is a simultaneous shortening of the dorsiflexors. This unloading can contribute to the reflex size (Nichols & Koffler-Smulevitz, 1991; Sinkjær et al., 1995).
4. Polysynaptic Group Ib and II reflex pathways appear to become operable during locomotion, and should in principle augment the stretch reflex responses (Gossard & Hultborn, 1991; Pearson et al., 1992).
5. When the soleus H- and the stretch reflex are conditioned by a tendon tap applied to the biceps femoris tendon, the H-reflex is strongly depressed (Morita et al., 1998). This inhibition is believed to be caused by presynaptic inhibition of the Ia afferents which mediate the reflex (Nielsen & Petersen, 1994). The stretch reflex is not depressed at all by the biceps femoris tendon tap. This can be explained by a lower sensitivity of the later part of the EPSP underlying the reflexes to presynaptic inhibition (Morita et al., 1998).

The mechanically strong stretch reflex found in the isometrically contracted ankle extensors during sitting (Allum & Mauritz, 1984; Toft et al., 1991) is generally believed to be diminished during the stance of walking, as implicated by the reduced H-reflex. However, the unchanged strength of the soleus stretch reflex suggests that the short latency afferent input from the stretched ankle extensors contributes as much to the ankle joint stiffness during walking as during sitting. This is consistent with recent experiments in which the stretch reflex showed a mechanically important contribution during simulated walking (Kearney et al., 1996).

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